# PRE-EXERCISE FEEDINGS OF GLUCOSE, FRUCTOSE, OR SUCROSE: EFFECTS ON FUEL HOMEOSTASIS IN RATS

by

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## Introduction

Interest among athletes to improve performance through dietary means has stimulated much research. Endurance athletes, in particular, are interested in prolonging optimal performance. One of the limiting factors in endurance exercise is the amount of stored glycogen. Several dietary factors can affect glycogen stores. For example, carbohydrate loading is known to increase glycogen stores, thereby increasing endurance (55). Carbohydrate loading is, however, accompanied by water retention and a feeling of heaviness, often considered undesirable.

Other methods of "sparing" glycogen stores through an increase in fat utilization have also been studied (13, 28, 19). An increase in the utilization of free fatty acids would, theoretically, spare the amount of carbohydrate used, thereby prolonging performance time.

One might anticipate that carbohydrate feedings prior to exercise would also spare glycogen stores. However, glucose feedings 30-45 minutes prior to exercise have been shown to have negative effects (9, 13, 22, 35). In these studies a rapid rise and fall in serum glucose upon initiation of exercise was observed, resulting in a reduction in exercise time to exhaustion. These effects may be due to the rapid rise in insulin also observed (35, 40, 9). This hyperinsulinemia is

followed by an exercise-induced decrease in blood glucose and greater muscle glycogen usage (48, 13).

Although the previous studies were performed using glucose, it is possible that other carbohydrates used in pre-exercise feedings may be preferred. Studies have shown a relatively low insulin response with fructose compared to glucose or sucrose (32, 15, 8, 35). Because insulin levels are lower for fructose one might expect reduced dependence on muscle glycogen. In addition, few studies have been done on sucrose, which is comprised of equimolar amounts of fructose and glucose. The purpose of this study was to compare the effects of various carbohydrates on blood metabolites and to see if different carbohydrates would, indeed, "spare" muscle glycogen.

## LITERATURE REVIEW

### I. Body Fuel Stores

The major energy stores in the body used during exercise are fatty acids and glucose from blood or muscle glycogen. To a lesser extent amino acids from body protein can also be used. These stores provide energy through the production of ATP, or adenosine triphosphate. The amount of ATP existing in the body at any given time is very small, and could provide maximal contraction for only a few seconds (45). However, it is continually being replaced through the breakdown of fat, glycogen, or creatine phosphate. The use of these substrates during exercise varies according to the intensity and duration of the exercise and the individual's level of training. Usually a mixture of two or more fuels is used during exercise, though one fuel may predominate. The available fuel sources in a 154-pound adult male are shown below (55).

| Source | Total Calories Available |
|---|---|
| 1. Muscle | |
| ATP | 1-2 |
| PC | 4-5 |
| Glycogen | 1500-1800 |
| Triglycerides | 2500-2800 |
| Protein | 25,000-30,000 |
| 2. Blood | |
| Glucose | 20-25 |
| Free Fatty Acids | 6-8 |
| Triglycerides | 70-80 |
| Proteins | 160-200 |
| 3. Adipose Tissue | |
| Triglycerides | 80,000-100,000 |
| 4. Liver | |
| Glycogen | 350-400 |

A. Creatine Phosphate. During high intensity anaerobic exercise of short duration (less than 30 seconds) such as sprinting or jumping, the body uses creatine phosphate (CP) for ATP resynthesis. The energy stored in the bonds of creatine phosphate are used to join phosphate with ADP, creating ATP and the by-product creatine. The cell's concentration of CP at rest is about 3-5 times greater than ATP and is considered the phosphate "reservoir." Creatine phosphate stores drop substantially after 2-3 minutes of heavy, vigorous exercise, accompanied by a paralleled decrease in ATP (5, 33, 24). It appears that the depletion of creatine phosphate is largely responsible, along with lactic acid accumulation, for fatigue after short, intense activity.

B. Glucose and Glycogen. Another reserve molecule that restores ATP during anaerobic exercise is glucose. Glucose may pass from the blood into the working muscle cell, or it can be supplied by glycogen stores within the muscle itself. This process is called anaerobic glycolysis and results in a net production of two ATP molecules and two molecules of lactic acid from one molecule of glucose. Accumulation of lactic acid is one factor contributing to fatigue. Lactic acid is an important source of energy that accumulates in the body during intense anaerobic activity. An advantage to this system is that it does not require oxygen and ATP can be produced fairly rapidly, but the exercise cannot be sustained for a long period

of time. Lactic acid accumulates and becomes available when activity is reduced and oxygen is present. Two hydrogen molecules in lactic acid are picked up by NAD, converting lactic acid back to pyruvic acid to continue through oxidative phosphorylation to yield 36 ATP.

If oxygen is readily available during submaximal exercise, 38 ATP can be released from the aerobic breakdown of one molecule of glucose. This results in a much greater production of ATP from glucose than if glucose were utilized anaerobically.

During high-intensity exercise, the prime contributor of energy is carbohydrate, primarily blood glucose and glycogen stores in muscle and liver. Blood glucose may supply as much as 30-40% of the total energy of the exercising muscles (21). During the initial stage of exercise, the glucose uptake by the muscles increases sharply and continues to increase, though at a slower rate as exercise progresses. After 40 minutes of exercise, the glucose uptake is 7-20 times greater than at rest. The high percentage of carbohydrate contribution is explained by the fact that under anaerobic conditions the supply of CP is limited and fatty acids must have oxygen for degradation.

During lower intensity continuous exercise, a somewhat greater amount of energy is derived from the breakdown of the body's stores of fat. About 40-50% of the energy requirement during submaximal exercise still comes from glycogen, but as

exercise continues and the glycogen stores are reduced, a greater percentage of the energy comes from the catabolism of fat.

C. Fat. Most of the dietary fat we consume is in the form of triglycerides and is metabolized immediately for energy or is stored primarily in adipose cells. Small amounts of triglycerides are also present in muscle and other tissues. These triglycerides undergo hydrolysis to provide three fatty acids and one glycerol molecule. The fatty acids are used by the exercising muscle. Free fatty acids released from fat cells are carried in the blood by plasma albumin to the muscle and are converted by beta-oxidation to acetyl CoA, which can then enter the citric acid cycle for energy production. Compared to 38 molecules of ATP formed from one molecule of glucose, 147 ATP's can be formed from the breakdown on an 18-carbon fatty acid molecule (stearic acid). A pound of fat can produce over twice as much ATP as a pound of pure carbohydrate, but more oxygen is required to break down the fat than is required for the breakdown of glucose. In aerobic exercise, carbohydrate is a more efficient fuel in terms of ATP produced per molecule of oxygen. However, because of the limited amount of carbohydrate that is stored in the body, individuals participating in long duration physical exercise rely more heavily on fat oxidation.

In long endurance activities, an increasingly large contribution of the fuel needed for the exercise comes from the

catabolism of fats (3, 11). Fats can supply 20% of the energy after one hour of continuous submaximal exercise, while after four hours of work, fat can supply up to 50% of the fuel supply. Increasing the availability of free fatty acids during long-term exercise would spare the muscle glycogen and blood glucose utilization rate in exercising muscle (47, 13, 28, 31, 19).

D. Proteins. Under normal conditions, proteins are not used for a substantial amount of energy during exercise. They are primarily used for maintenance, repair, and growth of body tissue, though some amino acids are catabolized during prolonged exercise. Through the process of transamination, the amino groups ($-NH_2$) from three branched chain amino acids, leucine, isoleucine, and valine, picked up by muscle are fixed onto pyruvate to make the amino acid alanine. Amino groups from glutamine may also be used to form alanine. Alanine is then released from exercising muscle and is circulated to the liver where it undergoes deamination and is converted to glucose. This is known as the alanine-glucose cycle. When amino acids are catabolized, their amino groups are converted to urea, which is excreted in the urine, and a carbon skeleton which may be used for energy or synthesis of other compounds.

The use of protein as an energy source has been reported to be 10-17% at rest (16, 39) and 4-15% during exercise ( 25, 36, 38, 51). Although the greater caloric expenditure during exercise would mean more total protein catabolism, the

percentages appear comparable. Thapar (53), Deutsch (17), and Konopka (39) have noted that significant amounts of urea are excreted in sweat during exercise. Konopka found no increase in urine urea, though serum urea significantly increased during exercise.

An important fact to consider in protein catabolism is the availability of other fuels. Lemon and Mullin (38) and Lemon (39) studied the effect of muscle glycogen stores on protein utilization during one hour of exercise. With high concentrations of muscle glycogen, protein catabolism was rather low, providing 4-6% of the total energy during exercise. However, when muscle glycogen levels were low, the energy contribution of protein rose to 10.4%. In another study, Konopka and Haynes (37) found that individuals having low levels of glycogen also exhibited greater losses of sweat urea nitrogen during exercise, indicating an inverse relationship between glycogen stores and protein utilization.

## II. Functions of Carbohydrates

Carbohydrates serve several important functions related to exercise performance, the most important being an energy source for the body. Simple and complex carbohydrates must be digested and converted to the simple 6-carbon sugar, glucose, before they can be used by the body. If not enough carbohydrates are ingested, glucose is then obtained from the catabolism of glycogen, a highly branched chain of linked glucose molecules stored in a limited supply in the liver and muscles, or from

catabolism of amino acids in body proteins. If an excess of carbohydrates are consumed in a meal, the carbohydrates can be converted to muscle glycogen and liver glycogen (storage). Once the capacity for glycogen storage is reached by the cell, the excess carbohydrate is then converted to fat and stored in adipose tissue.

Carbohydrates also provide a protein-sparing effect. Proteins, again, are primarily responsible for maintenance, repair, and growth of body tissues, but when a diet is low in carbohydrates, protein can be catabolized to synthesize glucose. This process, termed gluconeogenesis, provides the body with glucose when glycogen stores become depleted.

Another function of carbohydrates is to facilitate the metabolism of fat for energy. Fatty acid degradation via the Kreb's cycle requires oxaloacetic acid to combine with the acetyl CoA formed during beta-oxidation. The pyruvic acid formed during glucose metabolism may play an important role in furnishing this oxaloacetic acid. If supplies of carbohydrate are insufficient, either due to inadequate transport of glucose to the cell as in diabetes, depletion of glycogen through extreme exercise, or inadequate diet, the acetate fragments produced by beta-oxidation of fats accumulate in extracellular fluids. Without the carbohydrate-derived oxaloacetic acid, the acetyl-CoA cannot enter the Kreb cycle, and is instead converted to ketone bodies which leads to ketosis.

Finally, carbohydrate is necessary for proper functioning

of the central nervous system. Hypoglycemia, or low blood glucose, is characterized by weakness, hunger, and dizziness. Sustained and very low blood sugar can cause irreversible brain damage.

III. Factors Influencing Substrate Use

A. Hormones. The effects of insulin, glucagon and the catecholamines epinephrine and norepinephrine are probably the most important hormones involved in substrate availability during exercise (49, 29). Insulin is responsible for promoting glucose metabolism in the muscle, promoting fat synthesis, and inhibiting free fatty acid release. Glucagon basically exerts the opposite effects, stimulating glycogenolysis in the liver. High blood glucose stimulates insulin secretion by the pancreas, while low blood glucose stimulates glucagon secretion. Epinephrine has an effect on glucose metabolism similar to that of glucagon except that it stimulates breakdown of muscle glycogen as well as that in liver. During prolonged exercise when glucose levels fall, increases in cortisol have been noted. The major function of the glucocorticoid cortisol is to produce glucose from non-glucose precursors.

B. Intensity and Duration. One of the major factors determining substrate use is the supply of oxygen to the muscles, which is affected by exercise intensity. During high intensity anaerobic exercise, PC and muscle glycogen are the major energy substrates. Aerobic energy sources are a combination of

carbohydrates and fat metabolism. In general, however, the closer the exercise intensity is to the maximum level, the more carbohydrate is used as the energy substrate. More energy is derived per liter of oxygen consumed when carbohydrates are oxidized, giving carbohydrates an advantage over fat oxidation. However, body stores of carbohydrates are much less than those stores of fat, and can be a limiting factor in endurance exercise. Exercising at high intensity levels results in more carbohydrate metabolism and results in short duration; while low intensity exercises use more fat as an energy source and can be sustained for a long period of time.

C. Muscle Fiber Type. Muscles are comprised of several different types of muscle fibers, or cells. These muscle fibers can be one of three types: type Ia, type IIa, or type IIb. Type I fibers are red, slow twitch oxidative cells using primarily fat for energy. Type IIa fibers are red, fast twitch, oxidative glycolytic, using primarily carbohydrate for energy. Type IIb fibers are white, fast twitch, glycolytic, using mainly carbohydrates for energy. Fiber types, then, with their various abilities to utilize oxygen and different substrates are related to both the intensity and duration of exercise. The various muscles in the body may contain a majority of one muscle fiber or a mixture. A description of muscle fiber composition in individual rat muscles is shown in the table on the next page (2).

| Muscle | Muscle Fiber Type (%) | | |
|---|---|---|---|
| | IIa (red, fast) | IIb (white, fast) | I (red, slow) |
| Vastus Intermedius | 64 | 0 | 36 |
| Vatus Medialis | 53 | 46 | 1 |
| Vastus Lateralis | 56 | 42 | 2 |
| Lateral Gastrocnemius | 37 | 58 | 5 |
| Medial Gastrocnemius | 38 | 58 | 4 |
| Extensor Digitorum Longus | 59 | 38 | 3 |
| Flexor Digitorum Longus | 55 | 37 | 8 |
| Plantaris | 53 | 41 | 6 |
| Soleus | 16 | 0 | 84 |

D. Training Effects. Physiological effects due to training include an increased mitochondrial content in muscle, increased enzyme activity for oxidation of fat and glycogen, increased capillarization and increased stores of glycogen and triglycerides in the muscle (55). The ability to use more fat during exercise is due to increased levels of lipoprotein lipase and increased respiratory enzymes in the cell that are responsible for oxidation of free fatty acids. The increased fat utilization will spare the use of muscle glycogen and blood glucose.

## IV. Methods of Increasing Muscular Endurance

The ability to persevere in long endurance activities such as marathon running, cycling, or distance swimming is in part limited by the amount of glycogen that is stored in the working muscles prior to exercise (30, 6, 14). Methods of increasing muscular endurance include the following: 1) increasing the

amount of glycogen stored in the working muscles by increasing dietary carbohydrate several days prior to the exercise, or 2) sparing the amount of glycogen used for the given activity, i.e. by increasing fat catabolism or using glucose circulating in the blood. Although carbohydrate loading seems to be useful for many athletes, there are some undesirable side effects. Glycogen is stored bound with water, resulting in a weight gain of 1-2 kilograms. If a great deal of water is retained in the muscle, an uncomfortable, undesirable stiffness can result, as well as a heavier weight. Some might consider the retained water beneficial as it could help prevent dehydration and maintain blood volume when it is released during breakdown of glycogen. Finding methods to spare the use of glycogen in order to prolong the duration of the activity have included studying various carbohydrate feedings either before or during exercise.

A. Carbohydrates Before Exercise. Although a high carbohydrate diet in the days preceeding heavy exercise has shown a positive effect on endurance, as previously mentioned, other factors may influence the metabolism of carbohydrates ingested prior to exercise. These factors include the time between the ingestion of the carbohydrate and initiation of exercise, and the type of carbohydrate.

B. Time Factors. Carbohydrate ingestion 15-60 minutes prior to exercise will temporarily increase the blood glucose levels, decrease the blood free fatty acid levels and increase

the blood insulin levels. The combined effects of insulin and exercise can cause a decrease in blood glucose, resulting in hypoglycemia and fatigue. If, however, the carbohydrate is ingested immediately (1-5 minutes) prior to exercise, the effects were found to be different. Brooks (10) noted an increase in blood glucose when the sugar was ingested immediately before exercise. Luyckx (43) found the normal insulin response to glucose to be "blunted" when exercise immediately followed the carbohydrate intake.

C. Carbohydrates During Exercise. Glucose ingestion during intense exercise fails to increase serum concentrations of glucose and insulin (9). This is in contrast, however, with the rise in insulin observed when glucose is ingested during mild exercise (1). Norepinephrine responses during intense exercise are believed to inhibit the pancreatic release of insulin (4, 23).

D. Type of Carbohydrate. In 1981, Jenkins et. al. (32) developed a "glycemic index" in non-exercised subjects which showed the relative rate at which various foods raised blood glucose (Table 1). A high glycemic index means that carbohydrate from foods appears in the blood more rapidly than one with a low glycemic index. Some foods, such as sucrose and glucose resulted in a high index (fast release), while consumption of complex starches resulted in a lower index. Surprisingly, fructose also had a relatively low response. MacDonald (44) had already investigated the effects of various

TABLE 1
Glycemic index: the area under the blood glucose response curve for each food expressed as a percentage of the area after taking the same amount of carbohydrate as glucose (result are means of 5 to 10 individuals)

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Grain, cereal products | | | Vegetables | | | Fruit | | |
| Buckwheat | 51 ± 10* | (5) | Broad beans (25)¶ | 79 ± 16 | (6) | Apples (golden delicious) | 39 ± 3† | (6) |
| Bread (white) | 69 ± 5† | (10) | Frozen peas | 51 ± 6† | (6) | Banana | 62 ± 9* | (6) |
| Bread (whole-meal) | 72 ± 6† | (10) | Root Vegetables | | | Oranges | 40 ± 3† | (6) |
| Millet | 71 ± 10‡ | (5) | Beetroot (25)¶ | 64 ± 16 | (5) | Orange juice | 46 ± 6† | (6) |
| Pastry | 59 ± 6* | (5) | Carrots (25)¶ | 92 ± 20 | (5) | Raisins | 64 ± 11‡ | (6) |
| Rice (brown) | 66 ± 5† | (7) | Parsnips (25)¶ | 97 ± 19 | (5) | | | |
| Rice (white) | 72 ± 9§ | (7) | Potato (instant) | 80 ± 13 | (8) | Sugars | | |
| Spaghetti (wholemeal) | 42 ± 4† | (6) | Potato (new) | 70 ± 8* | (8) | Fructose | 20 ± 5† | (5) |
| Spaghetti (white) | 50 ± 8‖ | (6) | Potato (sweet) | 48 ± 6† | (5) | Glucose | 100 ± | (35) |
| Sponge cake | 46 ± 6† | (5) | Swede (25)¶ | 72 ± 8‡ | (5) | Maltose | 105 ± 12 | (6) |
| Sweetcorn | 59 ± 11§ | (5) | Yam | 51 ± 12§ | (5) | Sucrose | 59 ± 10§ | (5) |
| | | | Dried legumes | | | Dairy products | | |
| Breakfast cereals | | | Beans (tinned, baked) | 40 ± 3† | (7) | Ice cream | 36 ± 8' | (5) |
| All-Bran | 51 ± 5† | (6) | Beans (butter) | 36 ± 4† | (6) | Milk (skim) | 32 ± 5† | (6) |
| Cornflakes | 80 ± 6‡ | (6) | Beans (haricot) | 31 ± 6† | (6) | Milk (whole) | 34 ± 6† | (6) |
| Meusli | 66 ± 9§ | (6) | Beans (kidney) | 29 ± 8† | (6) | Yoghurt | 36 ± 4† | (5) |
| Porridge Oats | 49 ± 8‖ | (6) | Beans (soya) | 15 ± 5† | (7) | Miscellaneous | | |
| Shredded Wheat | 67 ± 10‡ | (6) | Beans (tinned, soya) | 14 ± 2† | (7) | Fish fingers | 38 ± 6† | (5) |
| Wheatabix | 75 ± 10‡ | (6) | Peas (blackeye) | 33 ± 4† | (6) | Honey | 87 ± 8 | (6) |
| | | | Peas (chick) | 36 ± 5† | (6) | Lucozade | 95 ± 10 | (5) |
| Biscuits | | | Peas (marrowfat) | 47 ± 3† | (6) | Mars bar | 68 ± 12‡ | (6) |
| Digestives | 59 ± 7* | (6) | Lentils | 29 ± 3† | (7) | Peanuts (25)¶ | 13 ± 6† | (5) |
| Oatmeal | 54 ± 4† | (6) | | | | Potato crisps | 51 ± 7† | (6) |
| Rich Tea | 55 ± 4† | (6) | | | | Sausages | 28 ± 6† | (5) |
| Ryvita | 69 ± 10‡ | (7) | | | | Tomato soup | 38 ± 9* | (5) |
| Water | 63 ± 9* | (6) | | | | | | |

Significance of difference from equivalent glucose load: * = $p < 0.01$; † = $p < 0.001$; ‡ = $p < 0.05$; § = $p < 0.02$; ‖ = $p < 0.002$; ¶ Only 25 g carbohydrate portion given. (32)

sugars on blood insulin and blood glucose response. The insulin response was greatest for glucose, one-third less for sucrose, and approximately 80% less for fructose. Koivisto (35) compared insulin responses to fructose and glucose. Fructose ingestion 45 minutes before exercise did not produce the hyperinsulinemia or hypoglycemia that was noted with the ingestion of glucose. Levine (40) found significantly reduced depletion of muscle glycogen during a 30 minute exercise period following ingestion of fructose than that for glucose or water. Higher insulin levels impede free fatty acid utilization for energy. Foster (22) reported that mobilization of lipids during exercise is impaired by prefeeding glucose and reduces performance. Hargreaves (26) found no differences in muscle glycogen between fructose and a sweet placebo though there was a trend for muscle glycogen to be lower when given fructose rather than glucose as a pre-exercise feeding. McMurray (46) compared glucose to fructose before high intensity endurance performance and found higher free fatty acids after fructose feedings which would allow more fat utilization. In all exercise studies cited above, the effects of pre-exercise fructose were not compared to sucrose, a more common and available carbohydrate. Sucrose is a dissacharide comprised of equimolar amounts of fructose and glucose. Based on its glycemic index value, which falls between glucose and fructose, it would theoretically result in effects, during exercise, that would fall between those of glucose and fructose. That is,

sucrose might elicit an insulin response between that of glucose and fructose, and also have similar glycogen-sparing effects. The comparison of these effects in rats fed either glucose, fructose, sucrose or unsupplemented water will be accomplished in the proposed study.

## MATERIALS AND METHODS

Animals and their care. One-hundred and ten male weanling Wistar rats (HSD:WI:BR, Harlan Sprague Dawley Inc., Indianapolis, IN) weighing 40-70 grams were allocated randomly to stainless steel cages in a temperature controlled room (21 C) which was maintained on a 12:12 hour light-dark cycle. The animals were fed Rodent Laboratory Chow (5001 Ralston Purina Co., St. Louis, MO) and water ad libitum throughout the entire seven week study. The nutritional content of the diet is shown in appendix table 1.

Experimental design. Four weeks after arrival, the animals weighing approximately 200 g were separated into seven exercise groups according to the planned seven days of sacrifice. There were 16 rats per group for the first six groups and the seventh group of "spare" rats was included in case some rats in the first six groups would not run.

Rats were trained on a zero-grade treadmill (Radiotrol Treadmill, 1/2 h.p., Boston Gears, Quincy, MA) two weeks prior to the planned day of sacrifice. A progressive exercise schedule was followed until animals could run comfortably at 18 m/min for 30 minutes. (See appendix table 2 for training program.)

Rats were then weighed the night before the trial (weights in appendix table 3) and were fasted overnight (12-16 hours). On the day of the trials they were force-fed a carbohydrate

solution 30 minutes before exercise. These solutions were 3 ml distilled water containing one of the following: 1) 2 g fructose, 2) 2 g glucose, 3) 2 g sucrose, or 4) no additional carbohydrate (control). When preparing carbohydrate solutions, it was found that carbohydrates differed in their solubility and volume displacement in water. In order to get 2 g of each carbohydrate in 3 ml water it was necessary to dissolve each of the following in 200 ml 85 C distilled water: 194 g fructose, 184 g glucose, and 166 g sucrose. Solutions were cooled to room temperature before force feeding. Carbohydrate content of each mixture was re-checked by drying 3 ml aliquots in a forced-air draft oven at 103 C for 4 hr and weighing after cooling in a desiccator.

Thirty minutes after receiving the pre-exercise feedings, animals were placed on the treadmill and exercised at 18 m/min. Within each dietary group, six rats were killed at each of the following time intervals: 0 (no exercise), 1,2, or 3 hours.

Sixteen rats, representing every dietary group and exercise time, were killed 15 minutes apart over four hours each day for six days. Diurnal variations among rats were reduced by randomizing the order of sacrifice each day while still killing one rat from each dietary group every hour and by killing between 11:30 a.m. and 3:30 p.m. each day. Those rats that would not run were eliminated and replaced on day seven at the appropriate time by another pre-trained rat. After exercising for the allotted period of time, rats were anesthetized with

sodium pentobarbitol (50 mg/kg) and ten ml of blood were drawn into a syringe, expressed into a tube, and centrifuged at 5000 g for 15 minutes. Serum was portioned in three separate vials and frozen at -18 C for later analysis of glucose, free fatty acids, and blood urea nitrogen. Muscles (soleus and red and white portions of the vastus lateralis) and liver (pyramidal lobe) were excised within ten minutes of death, weighed to the nearest .001 g, frozen in liquid nitrogen, and stored at -18 C until analysis for glycogen.

Analytical Procedures. Blood free fatty acids (FFA) were determined using a colorimetric micromethod based on the formation of FFA Cu soaps (50). Blood urea nitrogen was determined with a Sigma Kit (Sigma Kit No. 535, Sigma Chem. Company, St. Louis, MO). Blood glucose was also determined with a Sigma Kit (Sigma Kit No. 510). Muscle and liver glycogen were determined using the phenol-sulfuric acid method for small tissue samples after digesting the sample in NaOH and precipitating glycogen with ethanol (4). See appendix 4 for complete description of procedures for glycogen and FFA analyses.

Statistical Analysis. A graeco-latin square design with four dietary treatments, four exercise periods, and six rats for each diet/exercise combination was used. The means were adjusted and computed by the Least Squares Means option in SAS. See appendix table 5 for the computer program.

## RESULTS

Serum Glucose. After feeding rats a three ml carbohydrate solution containing glucose, sucrose, fructose or no additional carbohydrate (control), serum glucose levels were significantly higher ($P<0.05$) for all three carbohydrate groups than for the control group after 0,1,2, and 3 hours of running (Figure 1). At time 0 and after 2 hours of running, the sucrose-fed rats had a significantly higher ($P<0.05$) serum glucose level than did the glucose-fed rats and fructose-fed rats. No other significant differences were observed among rats fed the different carbohydrates.

Serum Free Fatty Acids. Free fatty acid levels in rat serum were not significantly different for any group at time 0 (Figure 2). However, after one hour of running, the water-fed control rats had significantly higher ($P<0.05$) FFA levels and this trend continued throughout the remainder of the three-hour trial. Few significant differences were noted when comparing FFA levels among the carbohydrate-fed rats. However FFA levels of glucose-fed rats tended to be the lowest and those of fructose-fed rats tended to be the highest. After two hours of running, the fructose-fed rats had significantly higher ($P<0.05$) FFA levels than did glucose-fed rats. The sucrose-fed rats tended to have FFA levels that were intermediate between those of the fructose-fed and glucose-fed rats.

Blood Urea Nitrogen. The blood urea nitrogen (BUN) levels for all dietary groups did not differ significantly at time 0

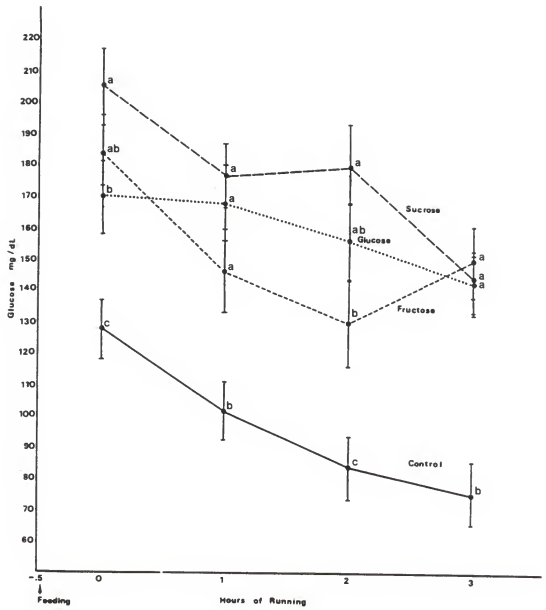


Figure 1-- Effect of pre-exercise carbohydrate feedings on serum glucose levels. Each value represents the mean ± standard error for six rats. Means at each time interval not sharing common superscripts differ ($P<0.05$).

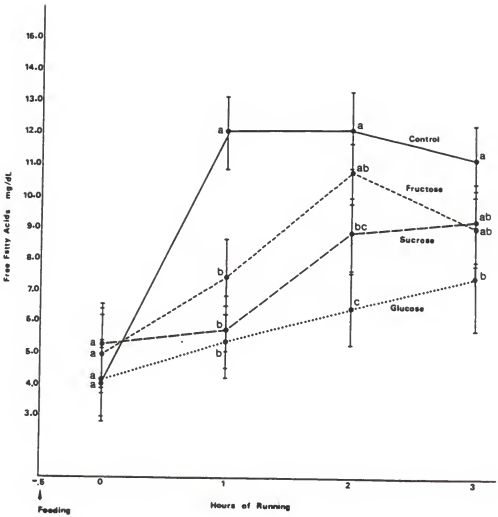


Figure 2-- Effect of pre-exercise carbohydrate feedings on serum free fatty acid levels. Each value represents the mean ± standard error for six rats. Means at each time interval not sharing common superscripts differ ($P<0.05$).

(Figure 3). After 2 hours of running, sucrose and glucose-fed rats had significantly lower ($P<0.05$) BUN levels than did control rats, while after 3 hours of running all carbohydrate-fed groups had significantly lower ($P<0.05$) BUN levels than control rats. After one hour of running, fructose-fed rats had significantly higher ($P<0.05$) BUN levels than sucrose-fed rats, and after 2 hours of running fructose-fed rats had significantly higher ($P<0.05$) BUN levels than both sucrose and glucose-fed rats. At 3 hours of running, however, the BUN levels of the fructose-fed rats were not significantly different from glucose or sucrose dietary groups.

Soleus Glycogen. Muscle glycogen contents of the soleus muscle are shown in Figure 4. Throughout the 3-hour study the water-fed control rats tended to have lower soleus glycogen contents than the carbohydrate-fed rats. At time 0, the fructose-fed rats had significantly less ($P<0.05$) soleus glycogen than did the glucose or sucrose-fed rats. After 1 hour of running, soleus glycogen in the fructose-fed group was still significantly less ($P<0.05$) than that of the sucrose-fed rats, but was not different when compared to that of the glucose-fed rats. After 2 hours of running there were no significant differences between any dietary group, and after 3 hours of running soleus glycogen in the fructose-fed rats was again significantly lower ($P<0.05$) than that of the glucose-fed rats.

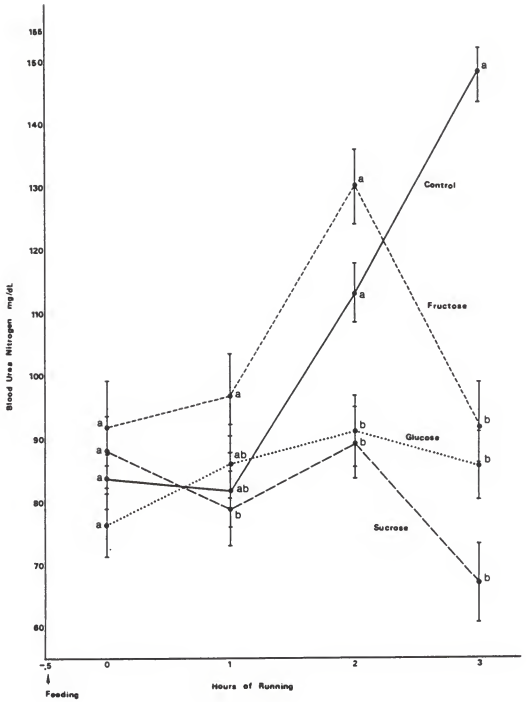


Figure 3-- Effect of pre-exercise carbohydrate feedings on blood urea nitrogen levels. Each value represents the mean ± standard error for six rats. Means at each time interval not sharing common superscripts differ ($P<0.05$).

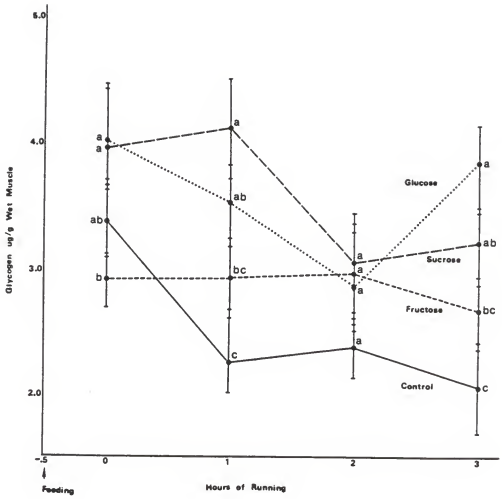


Figure 4-- Effect of pre-exercise carbohydrate feedings on muscle glycogen contents in the soleus muscle. Each value represents the mean $\pm$ standard error for six rats. Means at each time interval not sharing common superscripts differ ($P<0.05$).

Red Vastus Lateralis Glycogen. Again the water-fed control rats tended to have the lowest glycogen contents in the red vastus lateralis muscle, however, there were no significant differences noted between groups after 0,1, or 2 hours of running (Figure 5). After 3 hours of running, however, fructose and glucose-fed rats had a significantly higher ($P<0.05$) soleus glycogen contents than water-fed control rats; and glucose-fed rats had significantly more ($P<0.05$) soleus glycogen than sucrose-fed rats.

White Vastus Lateralis Glycogen. Few differences were noted when comparing white vastus lateralis muscle glycogen contents between groups during 3 hours of running. The water-fed control rats tended to have the lowest glycogen levels and after two hours of running the control rats had significantly less ($P<0.05$) muscle glycogen levels than did the sucrose-fed rats. No other differences were observed when comparing glycogen contents among rats fed the different carbohydrate solutions.

Liver Glycogen. The effect of pre-exercise carbohydrate feedings on liver glycogen contents are shown in Figure 7. After 0 and 3 hours of running, there were no significant differences in liver glycogen contents among any dietary group. Water-fed control rats tended to have the lowest liver glycogen levels throughout the 3-hour study. Fructose-fed rats after 1 hour of running had significantly less ($P<0.05$) liver glycogen

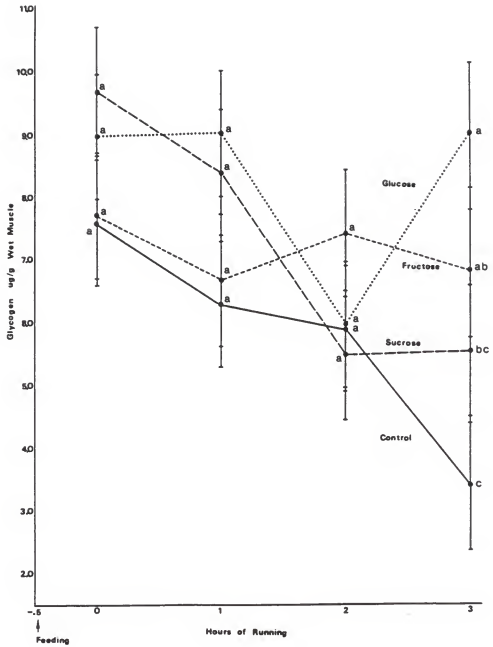


Figure 5-- Effect of pre-exercise carbohydrate feedings on glycogen contents in the red vastus lateralis muscle. Each value represents the mean ± standard error for five or six rats. Means at each time interval not sharing common superscripts differ ($P<0.05$).

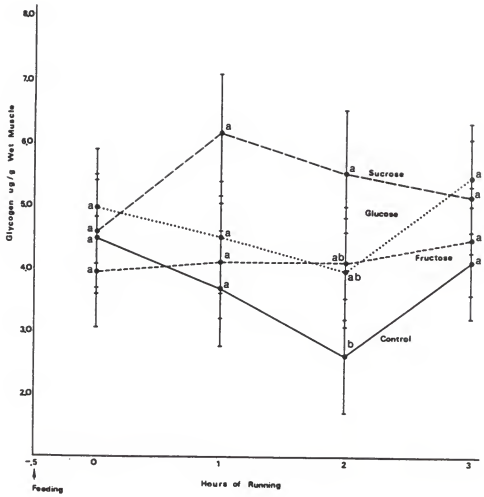


Figure 6-- Effect of pre-exercise carbohydrate feedings on glycogen contents in the white vastus lateralis muscle. Each value represents the mean ± standard error for six rats. Means at each time interval not sharing common superscripts differ ($P<0.05$).

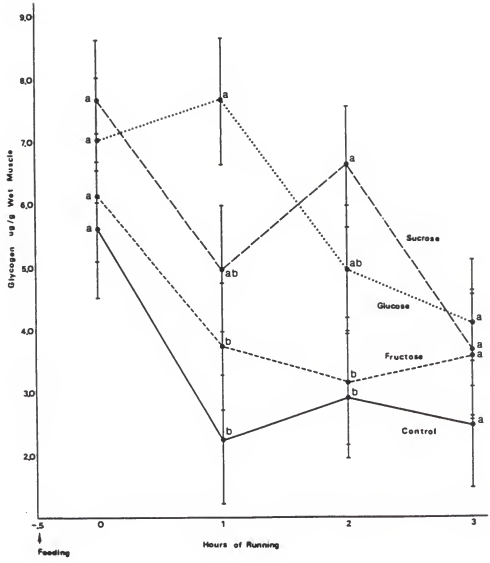


Figure 7-- Effect of pre-exercise carbohydrate feedings on liver glycogen contents. Each value represents the mean ± standard error for six rats. Means at each time interval not sharing common superscripts differ ($P<0.05$).

than did the glucose-fed rats, and after 2 hours of running fructose-fed rats had significantly less ($P<0.05$) liver glycogen than sucrose-fed rats. The numerical values for this study are shown in tables 2 and 3.

Table 2. Effect of pre-exercise carbohydrate feedings on free fatty acid, glucose, and blood urea nitrogen levels in rat serum. (Tabular values for Figures 1-3)*

| Pre-Exercise Feeding | Hours of Running | Free Fatty Acids | Glucose | Urea Nitrogen |
|---|---|---|---|---|
| | | mg/dl serum | | |
| Control | 0 | 3.89±1.16 | 127.4±11.1 | 82.4±7.7 |
| | 1 | 12.04±1.18 | 102.3±11.2 | 85.0±7.8 |
| | 2 | 12.29±1.18 | 86.5±11.2 | 114.6±7.8 |
| | 3 | 11.02±1.17 | 75.0±11.1 | 145.4±7.8 |
| Glucose | 0 | 4.00±1.18 | 169.3±11.2 | 77.4±7.8 |
| | 1 | 5.18±1.17 | 168.0±11.1 | 85.8±7.8 |
| | 2 | 6.31±1.16 | 155.3±11.1 | 89.9±7.7 |
| | 3 | 7.40±1.64 | 143.1±11.1 | 85.4±7.7 |
| Fructose | 0 | 4.84±1.17 | 181.3±11.1 | 91.7±7.8 |
| | 1 | 7.56±1.20 | 146.5±11.4 | 98.5±8.0 |
| | 2 | 10.65±1.20 | 132.5±11.4 | 130.1±8.0 |
| | 3 | 8.83±1.16 | 149.0±11.0 | 90.3±7.7 |
| Sucrose | 0 | 5.20±1.16 | 206.1±11.1 | 88.0±7.7 |
| | 1 | 5.55±1.20 | 176.8±11.4 | 74.7±8.0 |
| | 2 | 8.83±1.21 | 177.4±11.5 | 89.7±8.0 |
| | 3 | 9.36±1.18 | 147.6±11.2 | 71.0±7.8 |

*Adjusted mean ± standard error computed by the LSMeans option of SAS. Individual values are listed in appendix tables 6,7, and 8.

Table 3. Effect of pre-exercise carbohydrate feedings on muscle and liver glycogen levels. (Tabular values for Figures 4-7)*

| Pre-Exercise Feeding | Hours of Running | Red Vastus Lateralis | White Vastus Lateralis | Soleus | Liver |
|---|---|---|---|---|---|
| | | ug glycogen/g wet weight | | | |
| Control | 0 | 7.57±1.09 | 4.50±0.92 | 3.35±0.36 | 5.62±0.99 |
| | 1 | 6.31±1.01 | 3.71±0.93 | 2.25±0.37 | 2.26±1.00 |
| | 2 | 5.90±1.01 | 2.61±0.93 | 2.43±0.37 | 2.92±1.00 |
| | 3 | 3.43±1.00 | 4.11±0.93 | 2.01±0.36 | 2.47±0.99 |
| Glucose | 0 | 8.97±1.13 | 4.99±0.93 | 4.06±0.37 | 7.04±1.00 |
| | 1 | 9.00±1.00 | 4.50±0.92 | 3.51±0.36 | 7.67±0.99 |
| | 2 | 5.96±0.99 | 3.95±0.92 | 2.84±0.36 | 4.97±0.99 |
| | 3 | 9.15±0.99 | 5.49±0.92 | 3.85±0.36 | 4.04±0.99 |
| Fructose | 0 | 7.70±1.11 | 3.98±0.93 | 2.91±0.36 | 6.13±0.99 |
| | 1 | 6.67±1.15 | 4.12±0.95 | 2.98±0.37 | 3.71±1.02 |
| | 2 | 7.40±1.02 | 4.09±0.95 | 2.86±0.37 | 3.17±1.02 |
| | 3 | 6.78±1.09 | 4.47±0.92 | 2.62±0.36 | 3.53±0.99 |
| Sucrose | 0 | 9.67±0.99 | 4.59±0.92 | 3.97±0.36 | 7.66±0.99 |
| | 1 | 8.40±1.03 | 6.15±0.95 | 4.08±0.37 | 4.98±1.02 |
| | 2 | 5.48±1.13 | 5.51±0.96 | 3.01±0.38 | 6.66±1.03 |
| | 3 | 5.52±1.13 | 5.19±0.93 | 3.27±0.37 | 3.59±1.00 |

*Adjusted mean ± standard error computed by the LSMeans option of SAS. Individual values are listed in appendix tables 9, 10, 11, and 12.

## DISCUSSION

In this trial rats were used as a model to examine dietary treatments used by humans engaging in endurance exercise. The advantages for using rats are that their environment can be controlled, as well as their eating and exercise habits. However, one must recognize some shortcomings in conducting rat exercise trials before drawing inferences to humans.

It is possible that rats may metabolize carbohydrates differently than humans, however there is little research in this area to document this point. One problem encountered in the present trial was that there were some rats that refused to run on the treadmill despite continual prodding. Consequently, the variation observed in the measurements may, in part, reflect differences in the animals' preference for running. In spite of this problem, consistent trends were noted which support previous research and they will be presented.

This research was undertaken to examine the effects of pre-exercise feedings of different carbohydrates on available fuel sources in the body. One background theory regarding the ingestion of carbohydrates 30-60 minutes prior to exercise is that the carbohydrate will be metabolized and available as a source of energy when exercise begins. However, there is some evidence that pre-exercise feedings of carbohydrate may have adverse effects. The intake of high concentrations of glucose has been found to rapidly increase the blood glucose levels, causing a release of insulin which lowers blood glucose

resulting in a "rebound" hypoglycemia (1, 26, 35, 40). This hypoglycemia may be further enhanced by the onset of exercise (54). The insulin response also inhibits the release of free fatty acids, thereby causing more carbohydrate to be used for energy in the form of stored muscle or liver glycogen and available blood glucose. The pre-exercise muscle glycogen concentration is a major determinant of capacity for endurance exercise (7, 27, 34), so it is preferable to spare glycogen as much as possible.

In the present study the mean blood glucose level of water-fed control rats was lower than those of rats fed any of the carbohydrate solutions when measured 30 minutes after feeding and immediately prior to the onset of exercise. Blood glucose levels of the water-fed control rats also remained lower throughout the 3-hour exercise period. No "rebound" hypoglycemia was noted in any of the carbohydrate-fed animals when observed during this period.

The water-fed rats also generally exhibited the highest circulating levels of FFA suggesting that when rats are not fed carbohydrates prior to exercise, there is a greater reliance on fat as a fuel source during exercise. These findings corroborate those of Costill (13) and Dohm (19).

Also, the higher blood urea nitrogen (BUN) levels in water-fed rats suggest that a greater amount of amino acids may be used as fuel sources without pre-exercise carbohydrate feedings. It is well known that during exercise, certain amino

acids such as alanine are released from exercising muscle and are degraded by the liver to carbon skeletons (used for energy) and an amino group for incorporation into urea (20). Use of amino acids during exercise usually does not comprise a significant proportion of fuels utilized. However, this study suggests that pre-exercise feedings of either glucose, fructose, or sucrose may spare use of fatty acids and amino acids. A net loss of protein from muscle and liver following exhaustive exercise in the rat has been noted (18). One interesting observation in the present trial is that the carbohydrate-fed rats exhibited a reduction in BUN levels between two and three hours. The reason for this is not clear at this point.

Throughout the three hour exercise period the water-fed control rats also tended to have the lowest glycogen stores in their liver and in three muscles studied: the soleus, the red vastus lateralis, and the white vastus lateralis. The glycogen stores may have been low in those rats because they were 1) fasted for at least 12 hours overnight and 2) were also probably active the night before the trial (rats are nocturnal animals). The glycogen levels were not replenished in the water-fed control rats because they did not receive carbohydrates prior to exercise. Furthermore, glycogen levels tended to be higher in the carbohydrate-fed rats throughout exercise, suggesting that some replenishment had taken place, perhaps even within the 30-minute period prior to exercise.

One of the major objectives of this study was to see whether there was a difference in the available fuels when comparing rats fed different carbohydrate solutions 30 minutes prior to exercise. Jenkins (32) reported a lower insulin response and a slower, depressed rise in blood glucose when subjects were given fructose rather than sucrose or glucose. Hargreaves (26) found that subjects fed fructose 30 minutes before exercise exhibited a lower insulin response, more stable blood glucose levels, and higher post-exercise levels of muscle glycogen when compared to those fed glucose or water. These findings suggest that fructose ingestion may shift the fuel source from carbohydrate to fat and possibly lengthen exercise performance by sparing muscle and liver glycogen.

As the present study shows, fructose fed rats generally exhibited lower blood glucose levels than those fed either sucrose or glucose; and higher serum free fatty acid levels than those fed sucrose or glucose. Also, when comparing the effects of carbohydrate feedings, responses of fructose-fed rats were usually the closest to those of water-fed control rats. As seen by the circulating levels of fuels, perhaps fructose allows more utilization of fat than either of the other carbohydrate sources. If this is the case, then theoretically less carbohydrate will be used resulting in greater residual muscle and liver glycogen.

However, we did not observe a glycogen-sparing effect for fructose in this trial. In fact, the trend observed for

fructose-fed rats was that their glycogen levels in the soleus, red vastus, and white vastus muscles were initially lower than those of rats fed either glucose or sucrose-- up to 1 hour of exercise. One reason for the lower glycogen contents in fructose-fed rats is that the fructose-feeding may not have replenished the muscle glycogen as rapidly as did sucrose or glucose. As time of exercise progressed, the glycogen contents in all muscles of rats in the different dietary groups generally decreased.

Also, when observing liver glycogen contents, fructose-fed rats tended to have lower liver glycogen contents than those fed glucose or sucrose. In fact, throughout the 3-hour exercise trial, the fructose-fed rats did not have significantly different liver glycogen levels than those fed water only.

Another objective of this study was to determine whether sucrose would have effects that were intermediate between those of glucose and fructose, because it is comprised of equimolar amounts of these monosaccharides. From the variation observed in our animals, it is difficult to detect trends or patterns for sucrose-fed rats.

In summary, most of the differences observed in this trial were those between water-fed controls and those fed the carbohydrates. The water-fed control rats generally had lower blood glucose, higher blood free fatty acids and lower liver and muscle glycogen contents. This suggests that fasted

exercised rats rely more heavily on fat utilization during exercise than those fed carbohydrates 30 minutes prior to exercise.

A trend observed for fructose-fed rats was that their responses throughout the three-hour exercise trial were generally intermediate between those of water-fed rats and those of rats fed glucose or sucrose. This suggests that fructose-fed rats probably relied more on non-carbohydrate fuel sources during exercise than did the rats fed glucose or sucrose. No consistent trends or comparisons were noted for sucrose-fed rats.

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# APPENDIX

Appendix Table 1.

# Rodent Laboratory Chow 5001

## Description

Rodent Laboratory Chow® is a constant-formula rodent diet recommended for rats, mice and hamsters. The constant formula feature is designed to minimize nutritional variables in long-term studies. It is formulated for life-cycle nutrition; however, it is not designed for maximizing production in breeding colonies.

This product has been the standard of bio-medical research for approximately four decades.

**Features**

1 Constant formula

2 Formulated for multi-species
3 Standard rodent diet for biomedical research
4 Pellet or meal form

**Benefits**

1 Helps minimize nutritional variables that may affect your research
2 Single product in inventory
3 Scientific proof of product efficacy, experimental replication uses
4 Facilitates addition of test substances by eliminating grinding

## Guaranteed Analysis/Ingredients

| | |
|---|---|
| Protein, minimum | 23.0% |
| Fat, minimum | 4.5% |
| Fiber, maximum | 6.0% |

Ground extruded corn, soybean meal, ground oats, dried beet pulp, wheat germ meal, fish meal, brewers dried yeast, dehydrated alfalfa meal, cane molasses, dried milk products, meat and bone meal, wheat middlings, animal fat preserved with BHA, calcium carbonate, dicalcium phosphate, salt, calcium iodate, vitamin $B_{12}$ supplement, DL-methionine, calcium pantothenate, choline chloride, folic acid, riboflavin supplement, thiamin, niacin supplement, pyridoxine hydrochloride, ferrous sulfate, vitamin A supplement, D-activated animal sterol, vitamin E supplement, ferrous carbonate, manganous oxide, cobalt carbonate, copper sulfate, zinc sulfate, zinc oxide.

## Feeding Directions

Feed ad libitum to rodents. Plenty of fresh clean water should be available to the animals at all times.

RATS — Adult rats will eat 12 to 15 grams of diet per day. Feeders in rat cages should be designed to hold 2 to 3 days supply of feed at one time.

MICE — Adult mice will eat 4 to 5 grams of pelleted ration daily. Some of the larger strains may eat as much as 8 grams per day per animal. Feed should be available on a free-choice basis in wire feeders above the floor of the cage.

HAMSTERS — Adults will eat 10-14 grams per day.

## Chemical Composition[1]

| NUTRIENTS[2] | | Nitrogen-Free Extract | | Vitamins | |
|---|---|---|---|---|---|
| **Protein** % | 23.4 | (by difference) % | 49.0 | Carotene, ppm | 4.5 |
| Arginine % | 1.38 | **Gross Energy,** kCal/gm | 4.25 | Menadione (added), ppm | — |
| Cystine % | .32 | **Physiological Fuel Value[3],** kCal/gm | 3.30 | Thiamin, ppm | 17.7 |
| Glycine % | 1.20 | **Ash** % | 7.3 | Riboflavin, ppm | 8.0 |
| Histidine % | .55 | Calcium % | 1.00 | Niacin, ppm | 95.0 |
| Isoleucine % | 1.18 | Phosphorus % | .61 | Pantothenic Acid, ppm | 24.0 |
| Leucine % | 1.70 | Potassium % | 1.10 | Choline, ppm × 100 | 22.5 |
| Lysine % | 1.42 | Magnesium % | .21 | Folic Acid, ppm | 5.9 |
| Methionine % | .43 | Sodium % | .40 | Pyridoxine, ppm | 6.0 |
| Phenylalanine % | 1.03 | Chlorine % | .50 | Biotin, ppm | .07 |
| Tyrosine % | .68 | Fluorine, ppm | 35.0 | $B_{12}$, mcg/kg | 22.0 |
| Threonine % | .91 | Iron, ppm | 198.0 | Vitamin A, IU/gm | 15.0 |
| Tryptophan % | .29 | Zinc, ppm | 70.0 | Vitamin D, IU/gm | 4.5 |
| Valine % | 1.21 | Manganese, ppm | 64.3 | Alpha-tocopherol, IU/kg | 65.0 |
| **Fat** % | 4.5 | Copper, ppm | 18.0 | Ascorbic Acid, mg/gm | — |
| Cholesterol, ppm | 270.0 | Cobalt, ppm | 6 | | |
| **Fiber (Crude)** % | 5.8 | Iodine, ppm | 7 | | |
| Neutral Detergent Fiber[4] % | 16.0 | Chromium, ppm | 1.83 | | |
| Acid Detergent Fiber[5] % | 8.2 | Selenium, ppm | 20 | | |
| **Total Digestible Nutrients** % | 76.0 | | | | |

[1] Based on latest ingredient analysis information. Since nutrient composition of natural ingredients varies, analysis will differ accordingly.
[2] Nutrients expressed as percent of ration except where otherwise indicated. Moisture content is assumed to be 10.0% for the purposes of calculations.
[4] NDF = approximately cellulose, hemicellulose and lignin.
[5] ADF = approximately cellulose and lignin.
[3] Physiological Fuel Value (kCal/gm) = Sum of decimal fractions of protein (a) and carbohydrate. (Use Nitrogen-Free Extract × 4.9, 4 kCal/gm respectively.)

In: The Lab Facts Book published by the Ralston Purina Company, St. Louis, MO. p. 16.

Appendix Table 2. Rat training program (30 minutes/day) for two weeks prior to sacrifice.

| Day | Treadmill dial | Meters per minute |
|---|---|---|
| 1 | 25 | 6 |
| 2 | 35 | 10 |
| 3 | * | * |
| 4 | 40 | 12 |
| 5 | 45 | 14 |
| 6 | * | * |
| 7 | 50 | 16 |
| 8 | 55 | 18 |
| 9 | 55 | 18 |
| 10 | * | * |
| 11 | 55 | 18 |
| 12 | 55 | 18 |
| 13 | 55 | 18 |
| 14 | * | * |

*Rats rested on these days.
The experimental trials were conducted on day 15.

Appendix Table 3. Body weight of the experimental rats.*

| Hours of running | Pre-exercise feeding | | | |
|---|---|---|---|---|
| | Control | Glucose | Fructose | Sucrose |
| | g | | | |
| 0 | 347 | 386 | 336 | 375 |
| 0 | 381 | 338 | 372 | 341 |
| 0 | 335 | 356 | 358 | 318 |
| 0 | 310 | 346 | 318 | 347 |
| 0 | 350 | 341 | 331 | 325 |
| 0 | 312 | 322 | 319 | 319 |
| | 339±9** | 350±10 | 337±10 | 338±9 |
| 1 | 332 | 367 | 341 | 370 |
| 1 | 351 | 351 | 363 | 313 |
| 1 | 360 | 355 | 339 | 337 |
| 1 | 361 | 380 | 365 | 364 |
| 1 | 332 | 297 | 348 | 346 |
| 1 | 332 | 340 | 314 | 333 |
| | 342±10 | 348±9 | 345±10 | 346±10 |
| 2 | 377 | 346 | 363 | 375 |
| 2 | 367 | 330 | 367 | 356 |
| 2 | 331 | 366 | 339 | 328 |
| 2 | 383 | 340 | 336 | 367 |
| 2 | 294 | 331 | 340 | 359 |
| 2 | 345 | 322 | 331 | 365 |
| | 351±10 | 339±9 | 348±10 | 355±10 |
| 3 | 361 | 395 | 356 | 280 |
| 3 | 393 | 368 | 381 | 411 |
| 3 | 337 | 346 | 342 | 351 |
| 3 | 309 | 346 | 359 | 352 |
| 3 | 342 | 362 | 390 | 313 |
| 3 | 330 | 317 | 356 | 370 |
| | 347±9 | 355±9 | 364±9 | 346±10 |

*Body weight was measured the night before the exercise trial, prior to the fasting period. There were no significant differences between treatment means.
**Adjusted mean ± standard errror computed by the LSMeans option of SAS.

Appendix 4.

**ANALYTICAL PROCEDURES**

Free Fatty Acids

A colorimetric method using 2-(2-thiazolylazo-p-cresol) for the determination of FFA Cu soaps.

Reagents:

Extraction solvent: Chloroform heptane (1:1 v/v) containing 2% methanol, reagent grade.

Copper reagent: 10 ml 10 Ml aqueous $Cu(NO_3)_2$ and 5 ml of triethanolamine diluted with saturated sodium chloride solution to 100 ml. The pH is adjusted to 8.3 with 1 N sodium hydroxide.

TAC solution: 10 mg of 2-(2-thiazolylazo)-p-cresol (TAC) dissolved in 100 ml of ethanol and filtered.

Procedure:

1) 100 ul of serum was transferred to a glass test tube
2) 3.0 ml of extraction solvent was added
3) 1.0 ml of copper reagent was added
4) Tubes were immediately shaken mechanically for two minutes, and centrifuged at 5000 g for 20 minutes
5) 2 ml of the upper phase was transferred to another test tube
6) .5 ml of TAC solution was added and gently vortexed
7) The greenish blue color developed immediately and was measured at 610 nm against a reagent blank. The standard curve was prepared with palmitic acid.

Glycogen in tissue samples

Phenol-sulfuric acid reaction.

Reagents:

1) 30% potassium hydroxide saturated with sodium sulfate
2) 95% ethanol
3) 5% phenol (25 g phenol crystals dissolved in 500 ml distilled water)
4) Concentrated $H_2SO_4$
5) Standard glycogen solutions based on a 125 mg/25 ml stock standard solution.

Procedure:

Digestion

1) Frozen muscle (30-40 mg preferred) placed in glass test tube
2) .5 ml of 30% KOH solution was added to samples, making sure that the tissue was completely immersed in the solution
3) Tubes were placed in an 83 C water bath for 20-30 minutes until a homogenous solution was obtained
4) Tubes were then removed from the water bath and cooled on ice

Precipitation:

5) 95% ethanol (1.1-1.2 volumes) was added to precipitate the glycogen from the alkaline digest
6) Samples stood on ice for 30 minutes and were then

centrifuged at 5000 g for 20 minutes, the supernatants were aspirated

7) Glycogen precipitates were dissolved in 3 ml distilled water

8) An appropriate aliquot of the above solution was pipetted into glass test tubes and brought to a sample volume of 1 ml by addition of distilled water

| muscle | aliquot (ml) | distilled water (ml) |
|---|---|---|
| soleus | 0.2 | 0.8 |
| red vastus lateralis | 0.05 | 1.0 |
| white vastus lateralis | 0.5 | 0.5 |
| liver | 0.05 | 1.0 |

9) One milliliter of 5% phenol solution was added to the tubes

10) Five milliliters of 96-98% $H_2SO_4$ was added rapidly, the stream of acid being directed against the liquid surface rather than against the side of the test tube to ensure good mixing

11) The tubes were allowed to stand for 10 minutes, were vortexed, and were then placed in a 25-30 C water bath for 10-20 minutes

12) The absorbance was read at 435 nm against a reagent blank prepared with distilled water.All tests were carried out in triplicate.

Appendix Table 5.

```
//SUGARS  JOB (3910012>7,GFW=GE02),'ELIZABETH',TIME=(1,59)
/*REGION      500K
// EXEC SAS
//SYSIN DD *
DATA TRIALS;
INPUT (NUMBER FFA GLUCOSE BUN WEIGHT VASTUSR VASTUSW SOLEUS LIVER
   DIET KILLED START REP)(3. 3.2 2*3.1 4. 2*5.2 4.2 5.2 2*2. 2*1.);
CARDS;
$ADD EA_DATA
PROC PRINT;
PROC GLM;
CLASSES REP DIET START KILLED;
MODEL FFA GLUCOSE BUN WEIGHT VASTUSR VASTUSW SOLEUS LIVER=REP DIET
     START KILLED DIET*KILLED/E E& SS1;
LSMEANS DIET KILLED DIET*KILLED START/STDERR PDIFF;
/*
```

Appendix Table 6. Effect of pre-exercise carbohydrate feedings on serum glucose levels.

| Hours of running | Pre-exercise feeding | | | |
|---|---|---|---|---|
| | Control | Glucose | Fructose | Sucrose |
| | mg/dl | | | |
| 0 | 175.3 | 195.1 | 274.4 | 265.0 |
| 0 | 119.4 | 143.9 | 181.0 | 213.7 |
| 0 | 129.0 | 109.4 | 169.9 | 179.7 |
| 0 | 120.4 | 146.6 | 158.8 | 224.4 |
| 0 | 98.8 | 152.0 | 175.2 | 184.5 |
| 0 | 128.7 | 196.6 | 138.9 | 164.2 |
| | 127.4±11.1* | 169.3±11.2 | 181.3±11.1 | 206.1±11.1 |
| 1 | 196.5 | 170.0 | 201.3 | 219.1 |
| 1 | 85.5 | 129.0 | 156.2 | 176.9 |
| 1 | 69.7 | 185.6 | 114.1 | 167.0 |
| 1 | 83.2 | 158.6 | 121.2 | 153.6 |
| 1 | 70.7 | 180.3 | 141.7 | 195.6 |
| 1 | 109.1 | 184.9 | 142.1 | 149.9 |
| | 102.3±11.2 | 168.0±11.1 | 146.5±11.4 | 176.8±11.44 |
| 2 | 146.6 | 162.1 | 162.3 | 189.0 |
| 2 | 82.0 | 164.1 | 129.1 | 141.4 |
| 2 | 77.1 | 142.1 | 107.5 | 166.6 |
| 2 | 73.8 | 166.1 | 152.5 | 169.7 |
| 2 | 75.3 | 136.8 | 115.7 | 250.3 |
| 2 | 51.8 | 168.0 | 113.0 | 167.6 |
| | 86.5±11.2 | 155.3±11.05 | 132.5±11.4 | 177.4±11.5 |
| 3 | 82.3 | 106.4 | 186.0 | 233.2 |
| 3 | 71.4 | 147.8 | 133.4 | 97.2 |
| 3 | 91.8 | 118.4 | 123.8 | 149.7 |
| 3 | 67.7 | 178.7 | 187.7 | 152.4 |
| 3 | 75.9 | 144.2 | 166.1 | 109.1 |
| 3 | 65.1 | 168.3 | 104.2 | 127.6 |
| | 75.0±11.1 | 143.1±11.1 | 149.0±11.0 | 147.6±11.2 |

*Adjusted mean ± standard error computed by the LSMeans option of SAS.

Appendix Table 7. Effect of pre-exercise carbohydrate feedings on serum free fatty acid levels.

| Hours of running | Pre-exercise feeding | | | |
|---|---|---|---|---|
| | Control | Glucose | Fructose | Sucrose |
| | mg/dl | | | |
| 0 | 4.92 | 1.59 | 2.85 | 2.97 |
| 0 | 4.69 | 3.10 | 3.74 | 4.05 |
| 0 | 4.41 | 4.69 | 6.56 | 2.26 |
| 0 | 5.15 | 6.67 | 5.28 | 4.01 |
| 0 | 4.49 | 2.77 | 5.54 | 2.08 |
| 0 | 0.23 | 4.03 | 5.38 | 16.08 |
| | 3.89±1.16* | 4.00±1.18 | 4.84±1.17 | 5.20±1.16 |
| 1 | 12.38 | 3.41 | 5.77 | 4.74 |
| 1 | 10.90 | 4.82 | 8.49 | 8.15 |
| 1 | 12.64 | 6.26 | 8.23 | 5.72 |
| 1 | 10.05 | 5.77 | 8.26 | 7.67 |
| 1 | 15.03 | 5.13 | 7.62 | 4.10 |
| 1 | 11.05 | 6.51 | 5.38 | 3.90 |
| | 12.04±1.18 | 5.18±1.17 | 7.56±1.20 | 5.55±1.20 |
| 2 | 9.69 | 4.15 | 5.05 | 8.18 |
| 2 | 8.00 | 7.00 | 15.90 | 8.64 |
| 2 | 11.20 | 7.51 | 9.51 | 12.95 |
| 2 | 8.36 | 6.36 | 10.77 | 9.33 |
| 2 | 23.77 | 9.13 | 10.72 | 8.41 |
| 2 | 11.59 | 4.26 | 12.72 | 5.31 |
| | 12.29±1.18 | 6.31±1.16 | 10.65±1.20 | 8.83±1.21 |
| 3 | 13.59 | 10.23 | 8.28 | 3.59 |
| 3 | 9.59 | 5.38 | 9.13 | 13.10 |
| 3 | 8.77 | 8.44 | 10.13 | 9.10 |
| 3 | 13.28 | 6.90 | 10.72 | 7.56 |
| 3 | 11.61 | 7.74 | 10.44 | 10.92 |
| 3 | 10.08 | 5.49 | 4.85 | 10.82 |
| | 11.02±1.17 | 7.40±.1.64 | 8.83±1.16 | 9.36±1.18 |

*Adjusted mean ± standard error computed by the LSMeans option of SAS.

Appendix Table 8. Effect of pre-exercise carbohydrate feedings on blood urea nitrogen.

| Hours of running | Pre-exercise feeding | | | |
|---|---|---|---|---|
| | Control | Glucose | Fructose | Sucrose |
| | mg/dl | | | |
| 0 | 101.5 | 83.2 | 86.4 | 90.9 |
| 0 | 69.8 | 70.3 | 109.9 | 93.6 |
| 0 | 73.4 | 59.8 | 76.6 | 88.7 |
| 0 | 83.2 | 70.6 | 93.1 | 82.9 |
| 0 | 96.6 | 95.2 | 85.0 | 83.6 |
| 0 | 78.1 | 77.4 | 98.9 | 88.7 |
| | 82.4±7.7* | 77.4±7.8 | 91.7±7.8 | 88.0±7.7 |
| 1 | 68.6 | 92.5 | 94.6 | 71.3 |
| 1 | 71.7 | 77.3 | 84.2 | 69.3 |
| 1 | 76.3 | 87.9 | 91.3 | 108.7 |
| 1 | 108.1 | 83.2 | 102.9 | 60.8 |
| 1 | 76.9 | 97.7 | 99.6 | 80.4 |
| 1 | 92.7 | 76.6 | 110.2 | 81.9 |
| | 85.0±7.8 | 85.8±7.8 | 98.5±8.0 | 74.7±8.0 |
| 2 | 140.5 | 90.9 | 157.4 | 84.3 |
| 2 | 115.5 | 109.6 | 69.0 | 90.9 |
| 2 | 105.5 | 116.2 | 106.1 | 70.8 |
| 2 | 93.9 | 43.5 | 137.4 | 82.4 |
| 2 | 126.8 | 80.1 | 118.4 | 112.8 |
| 2 | 97.4 | 106.9 | 192.8 | 96.7 |
| | 114.6±7.8 | 89.9±7.7 | 130.1±8.0 | 89.7±8.0 |
| 3 | 145.8 | 105.7 | 80.6 | 41.9 |
| 3 | 143.1 | 64.2 | 65.2 | 101.1 |
| 3 | 161.0 | 77.5 | 83.8 | 72.5 |
| 3 | 160.1 | 61.0 | 125.4 | 43.0 |
| 3 | 142.5 | 88.2 | 120.6 | 85.5 |
| 3 | 135.7 | 115.7 | 74.4 | 58.1 |
| | 145.4±7.8 | 85.4±7.7 | 90.3±7.7 | 71.0±7.8 |

*Adjusted mean ± standard error computed by the LSMeans option of SAS.

Appendix Table 9. Effect of pre-exercise carbohydrate feedings on muscle glycogen contents in the rat soleus muscle.

| Hours of running | Pre-exercise feeding | | | |
|---|---|---|---|---|
| | Control | Glucose | Fructose | Sucrose |
| | ug glycogen/g wet weight | | | |
| 0 | 2.84 | 3.58 | 3.77 | 1.95 |
| 0 | 2.95 | 2.06 | 3.04 | 4.02 |
| 0 | 2.28 | 3.12 | 3.12 | 2.90 |
| 0 | 4.13 | 3.53 | 2.41 | 4.67 |
| 0 | 4.23 | 4.34 | 2.18 | 5.01 |
| 0 | 3.86 | 7.41 | 3.13 | 5.27 |
| | 3.35±.36* | 4.06±.37 | 2.91±.36 | 3.97±.36 |
| 1 | 1.91 | 1.84 | 2.47 | 3.58 |
| 1 | 1.82 | 3.05 | 3.17 | 3.95 |
| 1 | 2.01 | 4.84 | 2.04 | 4.67 |
| 1 | 2.79 | 5.24 | 3.02 | 5.13 |
| 1 | 2.47 | 3.51 | 3.96 | 4.93 |
| 1 | 2.51 | 2.75 | 2.89 | 2.39 |
| | 2.25±.37 | 3.51±.36 | 2.98±.37 | 4.08±.37 |
| 2 | 2.50 | 2.69 | 2.64 | 1.84 |
| 2 | 1.71 | 1.91 | 2.95 | 2.27 |
| 2 | 2.54 | 2.12 | 2.43 | 3.29 |
| 2 | 2.44 | 4.39 | 2.84 | 4.37 |
| 2 | 1.99 | 3.24 | 2.84 | 2.68 |
| 2 | 3.07 | 2.88 | 3.47 | 3.82 |
| | 2.43±.37 | 2.84±.36 | 2.86±.37 | 3.01±.38 |
| 3 | 2.11 | 2.20 | 2.07 | 3.04 |
| 3 | 1.69 | 4.52 | 2.03 | 1.86 |
| 3 | 2.05 | 2.73 | 3.24 | 2.89 |
| 3 | 1.86 | 5.69 | 2.18 | 5.31 |
| 3 | 2.42 | 3.70 | 3.62 | 2.40 |
| 3 | 2.06 | 4.29 | 2.74 | 3.82 |
| | 2.01±.36 | 3.85±.36 | 2.62±.36 | 3.27±.37 |

*Adjusted mean ± standard error computed by the LSMeans option of SAS.

Appendix Table 10. Effect of pre-exercise carbohydrate feedings on glycogen contents in the rat red vastus lateralis muscle.

| Hours of running | Pre-exercise feeding | | | |
|---|---|---|---|---|
| | Control | Glucose | Fructose | Sucrose |
| | ug glycogen/g wet weight | | | |
| 0 | 7.01 | 14.65 | 12.49 | 10.94 |
| 0 | 7.27 | 5.73 | 6.82 | 8.74 |
| 0 | -* | 10.42 | 8.20 | 10.88 |
| 0 | 8.60 | 9.12 | 8.63 | 9.70 |
| 0 | 6.10 | 8.36 | - | 7.16 |
| 0 | 8.18 | - | 2.09 | 10.33 |
| | 7.57±1.09** | 8.97±1.13 | 7.70±1.11 | 9.67±.99 |
| 1 | 2.63 | 4.88 | 8.09 | 3.32 |
| 1 | 7.85 | 6.93 | 9.41 | 10.92 |
| 1 | 7.30 | 13.41 | 5.33 | 6.10 |
| 1 | 7.17 | 10.12 | 6.70 | 9.40 |
| 1 | 7.05 | 7.16 | - | 8.93 |
| 1 | 6.08 | 10.31 | 8.20 | 10.18 |
| | 6.31±1.01 | 9.0±1.00 | 6.67±1.15 | 8.40±1.03 |
| 2 | 5.76 | 7.02 | 9.69 | 2.52 |
| 2 | 6.65 | 7.23 | 7.61 | 5.40 |
| 2 | 6.33 | 2.96 | 9.87 | 6.97 |
| 2 | 7.81 | 8.93 | 5.82 | 6.85 |
| 2 | 3.90 | 6.42 | 5.51 | - |
| 2 | 7.11 | 2.14 | 5.17 | 6.99 |
| | 5.90±1.01 | 5.96±.99 | 7.40±1.02 | 5.48±1.13 |
| 3 | 1.45 | 10.09 | 9.66 | 4.55 |
| 3 | 2.96 | 11.86 | 6.88 | 3.90 |
| 3 | 2.17 | 7.51 | 3.01 | 10.19 |
| 3 | 6.62 | 12.98 | 8.54 | 9.90 |
| 3 | 3.08 | 6.99 | - | 2.25 |
| 3 | 3.00 | 5.74 | 6.65 | * |
| | 3.43±1.00 | 9.15±.99 | 6.78±1.09 | 5.52±1.13 |

*These samples were lost.

**Adjusted mean ± standard error computed by the LSMeans option of SAS.

Appendix Table 11. Effect of pre-exercise carbohydrate feedings on glycogen contents in the rat white vastus lateralis muscle.

| Hours of running | Pre-exercise feeding | | | |
|---|---|---|---|---|
| | Control | Glucose | Fructose | Sucrose |
| | ug glycogen/g wet weight | | | |
| 0 | 7.23 | 4.67 | 1.39 | 6.91 |
| 0 | 4.42 | 4.83 | 4.99 | 5.11 |
| 0 | 2.92 | 4.87 | 8.50 | 2.83 |
| 0 | 3.62 | 7.20 | 5.18 | 5.68 |
| 0 | 0.78 | 6.45 | 0.94 | 3.92 |
| 0 | 7.37 | 3.98 | 1.13 | 3.45 |
| | 4.50±.92* | 4.99±.93 | 3.98±.93 | 4.59±.92 |
| 1 | 2.46 | 1.99 | 3.72 | 2.25 |
| 1 | 6.31 | 2.12 | 2.82 | 6.31 |
| 1 | 1.63 | 3.85 | 2.12 | 7.58 |
| 1 | 3.57 | 9.29 | 6.79 | 9.87 |
| 1 | 4.27 | 7.07 | 3.15 | 7.36 |
| 1 | 2.11 | 1.61 | 7.44 | 5.16 |
| | 3.71±.93 | 4.50±.92 | 4.12±.95 | 6.15±.95 |
| 2 | 2.91 | 2.05 | 2.48 | 0.59 |
| 2 | 5.15 | 5.83 | 8.35 | 2.98 |
| 2 | 3.20 | 1.65 | 2.75 | 1.80 |
| 2 | 1.59 | 6.87 | 5.31 | 11.05 |
| 2 | 2.85 | 3.01 | 2.10 | 9.32 |
| 2 | 2.03 | 3.64 | 4.62 | 4.81 |
| | 2.61±.93 | 3.95±.92 | 4.09±.95 | 5.51±.96 |
| 3 | 4.65 | 4.54 | 3.47 | 8.36 |
| 3 | 5.02 | 5.28 | 4.71 | 3.58 |
| 3 | 1.19 | 4.91 | 7.72 | 6.84 |
| 3 | 5.28 | 6.58 | 4.45 | 2.82 |
| 3 | 6.75 | 5.70 | 2.94 | 7.27 |
| 3 | 2.25 | 5.54 | 2.91 | 2.76 |
| | 4.11±.93 | 5.49±.92 | 4.47±.92 | 5.19±.93 |

*Adjusted mean ± standard error computed by the LSMeans option of SAS.

Appendix Table 12. Effect of pre-exercise carbohydrate feedings on liver glycogen contents.

| Hours of running | Pre-exercise feeding | | | |
|---|---|---|---|---|
| | Control | Glucose | Fructose | Sucrose |
| | ug glycogen/g wet weight | | | |
| 0 | 2.43 | 8.91 | 9.17 | 11.07 |
| 0 | 7.52 | 5.69 | 4.35 | 2.87 |
| 0 | 3.73 | 4.19 | 9.50 | 7.40 |
| 0 | 4.44 | 7.27 | 5.03 | 7.19 |
| 0 | 6.98 | 5.85 | 8.47 | 6.43 |
| 0 | 8.73 | 9.51 | 1.67 | 10.31 |
| | 5.62±.99* | 7.04±1.00 | 6.13±.99 | 7.66±.99 |
| 1 | 2.61 | 5.43 | 3.59 | 4.52 |
| 1 | 2.43 | 6.68 | 4.94 | 2.21 |
| 1 | 2.94 | 11.78 | 4.64 | 2.82 |
| 1 | 2.50 | 12.95 | 3.06 | 6.94 |
| 1 | 2.75 | 1.39 | 3.09 | 6.63 |
| 1 | 2.39 | 7.88 | 3.44 | 4.15 |
| | 2.26±1.00 | 7.67±.99 | 3.71±1.02 | 4.98±1.02 |
| 2 | 2.31 | 2.88 | 4.92 | 2.38 |
| 2 | 2.99 | 4.27 | 3.24 | 7.55 |
| 2 | 2.50 | 2.28 | 3.01 | 12.64 |
| 2 | 1.86 | 5.04 | 1.51 | 8.26 |
| 2 | 3.15 | 2.48 | 1.78 | 7.98 |
| 2 | 3.89 | 12.96 | 2.55 | 3.93 |
| | 2.92±1.00 | 4.97±.99 | 3.17±1.02 | 6.66±1.03 |
| 3 | 1.32 | 3.06 | 5.77 | 2.83 |
| 3 | 3.88 | 5.28 | 3.25 | 2.94 |
| 3 | 2.12 | 4.03 | 2.79 | 5.28 |
| 3 | 2.51 | 7.51 | 3.08 | 4.20 |
| 3 | 1.65 | 2.31 | 1.90 | 2.12 |
| 3 | 2.11 | 2.72 | 4.47 | 4.68 |
| | 2.47±.99 | 4.04±.99 | 3.53±.99 | 3.59±1.00 |

*Adjusted mean ± standard error computed by the LSMeans option of SAS.

# PRE-EXERCISE FEEDINGS OF GLUCOSE, FRUCTOSE, OR SUCROSE: EFFECTS ON FUEL HOMEOSTASIS IN RATS

by

ELIZABETH ELAINE ADDINGTON

B.S., Kansas State University, 1984

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AN ABSTRACT OF A MASTER'S THESIS

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ABSTRACT

The objective of this study was to compare the effects of various carbohydrate feedings on fuel stores during a three-hour bout of exercise in trained rats. Ninety-six rats were trained to run on a treadmill for 2 weeks. They were then divided into four dietary groups, fasted overnight, and fed three ml of a solution of water only, or water containing 2 g fructose, glucose, or sucrose 30 minutes prior to exercise. Six rats from each dietary group were killed after 0,1,2 or 3 hours of exercise. Blood glucose, free fatty acids, and blood urea nitrogen levels were measured as well as glycogen contents of the liver and three muscles including the soleus, red and white portions of the vastus lateralis. Carbohydrate-fed rats generally had higher levels of muscle and liver glycogen, higher blood glucose levels and lower levels of free fatty acids than control rats. These data suggest that pre-exercise feedings of carbohydrates generally increase body sources of carbohydrate for energy available during exercise. As shown by the lower free fatty acid levels in these animals, carbohydrate feeding may also reduce reliance on fat oxidation during exercise.

Of the carbohydrates studied, fructose feeding seemed to have the least impact on blood glucose levels and muscle glycogen, particularly during the early exercise period. Because fructose-fed rats also tended to have higher free fatty

acid levels than those fed sucrose or glucose, data suggest that pre-exercise feedings of fructose result in a greater reliance on fat for exercise energy than when the other carbohydrates were tested.